CHAPTER 6

Arthralgia

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According to the National Center for Health Statistics' report on health in the United States published in 2009, arthritis and musculoskeletal conditions were the most common causes of limitation of work among the working-age population. Of all symptom-based visits to physician offices, musculoskeletal complaints are the most common presenting symptom, ahead of respiratory and psychological complaints, which ranked second and third, respectively.

The cardinal signs of inflammation, *rubor* or redness, *calor* or heat, *dolor* or pain, and tumor or swelling, are used as examination findings to support the diagnosis of arthritis rather than arthralgia. Redness and heat are generally seen with inflammation rather than with joint and connective tissue disease. Limitation of passive range of motion (ROM) versus active ROM may indicate joint effusion, which is seen with intra-articular disease—traumatic, inflammatory, or degenerative. Tenderness over bony surfaces raises the concern for fracture and should generally be evaluated radiologically.

SEPTIC JOINT

Joint infection is an orthopedic emergency because untreated joint infection can lead to permanent cartilage loss and ultimate joint dysfunction. Synovial tissue has no basement membrane, so bacteria can easily enter a joint and cause infection. Acute monoarthritis has a short differential diagnosis, primarily crystal arthropathy and infection. At times patients with infectious arthritis exhibit other symptoms of infection, such as fever (64%), malaise, or a primary source of infection, so joint fluid analysis is crucial to the diagnosis of an acute monoarthritis.

Infectious arthritis has three main patterns: acute monoarthritis with common organisms such as *Staphylococcus aureus;* gonococcal arthritis, which is typically polyarticular and involves small joints; and chronic infectious, which can involve large and small joints and may be migratory (i.e., Lyme disease). The knee is the most commonly affected joint. There is a high chance of permanent loss of joint function with acute infectious monoarticular arthritis and up to a 15% mortality rate, thus appropriate diagnosis and management are crucial.

Management of a septic joint typically involves either surgery, antibiotics, or both. The surgical approach can be open or arthroscopic or can involve repeated joint aspirations to remove infected fluid as

it accumulates. Most practitioners advocate an approach of "septic joint until proven otherwise" when approaching an acute monoarticular arthritis.

Symptoms

- Intense joint pain +++++ (this does not differentiate infectious from other forms of arthritis)
- New joint swelling ++++
- Sweats, rigors +
- Prosthetic joint +

Signs

- Fever (50%)
- Joint warmth, tenderness, and decreased ROM

Workup

- Joint aspiration with microscopic examination, crystal analysis, and culture. Joint fluid WBC count greater than 100,000 per cubic milliliter should be considered infectious until proven otherwise. Fluid may be turbid with decreased stringing.
- CBC with WBC greater than 10,000/μL
- Elevated ESR
- Elevated CRP
- Blood culture (positive in 50%)
- Consider testing for gonorrhea by throat culture or urine, urethral, or cervical sample.

Comments and Treatment Considerations

Immediate surgical consultation is indicated to assist in decision making when a septic joint is suspected. Antibiotic coverage for *S. aureus, Streptococcus pneumoniae,* and gram-negative bacilli is generally indicated, in the form of a third-generation cephalosporin or fluoroquinolone in the case of cephalosporin allergy. Methicillin-resistant *S. aureus* (MRSA) has become an increasingly common community-acquired organism, so vancomycin may be added if this is a concern. Antibiotics may be required for 2 to 4 weeks followed by an additional 2 to 6 weeks of oral antibiotics. Coverage may be narrowed once an organism has been identified.

Surgical approaches may include arthroscopic drainage and lavage or open drainage and lavage. Drain tubes may be placed to allow persistent accumulation of pus to be removed. Serial arthrocentesis can also allow for joint fluid to be removed, but this approach does not allow for joint lavage.

Despite hospitalization, surgery, and antibiotic use, mortality from joint sepsis may be up to 15%. Immediate treatment after a high level of suspicion may help mitigate this reality.

GOUT

Gout is classically described as an acute monoarthritis, with a propensity for the first metatarsophalangeal (MTP) joint and foot in general,

the knee, and sometimes the hands, wrists, and elbows. Acute gout is clinically indistinguishable from pseudogout in which calcium pyrophosphate, rather than uric acid, is the offending crystallized solute. Gout has an intercritical form in which uric acid can have other negative systemic effects, such as nephrolithiasis. Chronic gout is a destructive joint disease potentially causing significant disability.

Gout is the result of the inflammatory effect within the joint stimulated by the presence of precipitated uric acid crystals. The influx of neutrophils, releasing nonspecific inflammatory products, is the culprit in the intense inflammation and not the "needle-like" effect of the crystals themselves. Hyperuricemia raises the likelihood of a gout attack, but elevated levels of uric acid are not necessary to make the diagnosis of gout. Men are more often affected than women. Obesity, renal insufficiency, thiazide diuretic use, cyclosporine therapy, high purine intake, excess alcohol intake, and family history are also predisposing factors.

Symptoms

- Joint pain that is intense +++++ Patients often report that the lightest touch on the joint is excruciatingly painful.
- Fairly rapid onset of joint pain ++++
- Swelling and redness +++++
- Often the classic gout joints, such as the first MTP or knee affected ++++
- Chronic gout may include ongoing joint pain because of erosions of bone and joint space caused by inflammation. +
- Tophi are usually painless, developing insidiously and without inflammation. ++

Signs

- Usually one joint affected +++++
- Redness and synovitis around the affected joint. The appearance may resemble acute cellulitis. +++++
- Severe tenderness to palpation +++++
- Limited active and passive ROM ++++
- Chronic gout may have tophi, which are firm, nontender nodules, generally over joints affected and often on extensor surfaces. ++

Workup

- Look for signs of chronic gout.
- Joint aspiration with fluid analysis should be performed if possible, at least in first occurrences, to help in distinguishing from joint infection. Joint fluid findings should include negatively birefringent crystals, greater than 2000 but less than 100,000 WBCs/mL, absence of Gram-staining organisms, and negative fluid culture. Viscosity of fluid is typically low, as is fluid "stringing." +++++
- Serum uric acid may be measured, but normal levels do not rule out gout. A serum level of 9 mg/dL is associated with a 4.9% annual incidence in healthy men, compared with 0.1% annual incidence for a level of less than 7 mg/dL.

- Evaluation for predisposing factors such as hypertension, renal insufficiency ++
- Consider evaluation for insulin resistance as a factor influencing uric acid excretion and as a risk factor for heart disease. + Hyperuricemia is a documented risk factor for cardiovascular disease as well.
- Young age at first presentation (20s or 30s) may warrant evaluation for inheritable enzyme deficiencies like hypoxanthine guanine phosphoribosyltransferase. +

Comments and Treatment Considerations

Decrease pain and inflammation with NSAIDS, noting that there is no proven advantage of any one NSAID. Ibuprofen derivatives, naproxen, sulindac, indomethacin, and cyclooxygenase-2 (COX-2) inhibitors have all shown similar efficacy (e.g., naproxen sodium 500 mg twice a day for 3 days then 250 mg twice daily until resolution).

Note that all NSAIDs (including COX-2 inhibitors) carry a black box warning about increase in acute cardiovascular risk. Hyperuricemia is a risk factor for cardiovascular disease (CVD).

Systemic (either by mouth or IM) or intra-articular corticosteroids are effective, provided that joint sepsis is not a consideration (e.g., prednisone 60 mg by mouth once daily for 3 days then taper).

Oral colchicine may be started at 0.6 mg by mouth up to three doses then dosing 0.6 mg one every other day to twice daily for prophylaxis from further attacks. Colchicine is renally excreted and must be dosed based on creatinine clearance. Patients with creatinine clearance less than 10 mL/min should not receive colchicine. Side effects of diarrhea, nausea, and vomiting are common. Colchicine is most effective if started on the first day of an attack.

For chronic and suppressive conditions, lifestyle modifications such as weight loss and decreased alcohol intake (particularly beer) are important. Diets lower in carbohydrate with proportional increase in unsaturated fat and protein have been proven effective in lowering serum uric acid levels.

Discontinuation of thiazide diuretics and consideration of discontinuation of cyclosporine, if possible.

When colchicine is used for long-term prophylaxis, patients should be monitored for muscle weakness (myopathy caused by the drug), bone marrow suppression, and renal insufficiency. Colchicine or an NSAID should be started concurrently with allopurinol or a uricosuric to prevent the precipitation of a gout episode in the first 3 to 4 weeks of therapy.

Allopurinol is effective both for underexcreters (75% to 90% of gout sufferers) and overproducers (10% to 25% of gout sufferers). Allopurinol should not be started within 3 to 4 weeks of an acute attack because sudden changes in serum uric acid levels during this period may stimulate an attack. Two percent of patients may develop an allergic reaction to allopurinol, which is generally mild, although severe allergic reaction is possible. Dosing should be started at 100 mg daily and increased until serum uric

acid concentrations are normalized (30% reduction in episodes if serum uric acid is 4.6 to 6.6 mg/dL). The usual daily dose is 200 to 300 mg/day. Allopurinol should be dosed based on the creatinine clearance.

Uricosurics have shown similar efficacy to allopurinol for underexcreters of uric acid in a prospective parallel study. Uric acid excretion should be documented at less than 700 mg per 24 hours. Probenecid is started at 250 mg by mouth twice daily and is increased up to 2000 mg daily in divided doses to lower serum uric acid level to less than 6 mg/dL. Sulfinpyrazone dosing starts at 50 mg by mouth twice daily and can be increased to 400 mg daily in divided doses, again with a target serum uric acid level of less than 6 mg/dL. Creatinine clearance must be monitored and uricosuric use can precipitate urolithiasis.

Uric acid oxidase is used for gout treatment and prevention in patients undergoing chemotherapy. A case study has demonstrated efficacy of tumor necrosis factor (TNF) blockade (etanercept) in treatment of refractory tophaceous gout. A novel xanthine oxidase inhibitor, febuxostat, has been shown more effective than allopurinol in a head-to-head trial.

HIP FRACTURE

Hip fracture remains a harbinger of poor outcomes in older adult patients. A 2000 report on fall prevention in older adults, notes, "Falls are the leading cause of injury deaths and disabilities among persons aged older than 65 years," and 95% of hip fractures are caused by falls.

Risks for falling are familiar territory to family physicians in working with older adult patients and include:

- Physical inactivity leading to loss of strength and balance
- Difficulty completing activities of daily living
- The presence of dementia
- Use of medications that cause cognitive impairment
- Home hazards such as poor lighting and throw rugs
- Having more than one chronic disease
- History of stroke
- Parkinson's disease
- Neuromuscular disease
- Urinary incontinence
- Visual impairment

Many of these risk factors are modifiable through intervention in the home. Interventions that Cochrane calls "likely to be beneficial" include multidisciplinary home evaluation and intervention in patients with a defined history of falls, professionally led strengthening and balance training, professional home hazard modification, withdrawal of psychotropics, cardiac pacing when indicated, and tai chi. The benefit of other interventions is less clear.

Treatment and prevention of osteoporosis using calcium and vitamin D supplementation and weightbearing exercise earlier in life,

bisphosphonates, calcitonin, and raloxifene all help to increase bone density and have proved fracture reduction. Calcium and vitamin D supplementation after menopause has been historically recommended, though this recommendation has been called into question. There is some apparent benefit of vitamin D alone in preventing falls in nursing home patients.

Symptoms
- Typically follow a fall ++++
- Pain in the area often with and without movement +++++
- It is not uncommon that patients walk on the injured leg for some time before presenting for care.

Signs
- Limitation of active and passive ROM secondary to pain +++++
- Shortening, internal rotation and abduction of the affected hip ++++

Workup
- Plain x-ray: A posteroanterior (PA) view of the pelvis and lateral view of the femur +++++
- MRI (or CT if MRI is unavailable or contraindicated) can be used if a high index of suspicion is present and the fracture is not apparent on plain x-ray.
- Hip fracture can occur at the femoral neck, between the greater and lesser trochanter (intertrochanteric), or below the lesser trochanter. The third is the least common type.

Comments and Treatment Considerations
Early surgical repair within 24 to 48 hours is optimal. If surgical risk assessment needs to be done prior to the procedure, outcomes are better if this is done in less than 72 hours. Displaced femoral neck fractures treated with primary arthroplasty result in similar mortality but better function and fewer surgery-related complications.

Most patients undergoing hip fracture repair should be treated with thromboprophylaxis with unfractionated heparin, low-molecular-weight heparin (LMWH), direct thrombin inhibitors, or warfarin. If anticoagulation is contraindicated, antithrombotic pumps are also effective in preventing deep vein thrombosis (DVT). Anticoagulation should be continued for 10 to 14 days after surgery and longer if the patient has risk factors for DVT such as inability to ambulate, obesity, malignancy, history of DVT, and older age.

Patients should be mobilized as early after fracture repair as possible. Antibiotics during surgery reduce the incidence of infection. Pain control is important in the management of any operative procedure. Addressing hospital- or surgery-associated delirium in older adult patients is important in preventing perioperative complications of repeat falling and patient distress.

KNEE AND ANKLE TRAUMA

Knee and ankle trauma are two of the most common concerns that bring patients to a family physician office. Family physicians should recognize and appropriately manage these injuries as they present to the office. The patient's story will give pertinent clues to lead to the appropriate diagnosis. Often the injury can be intuited from the mechanism of injury that the patient reports.

ANKLE TRAUMA

Common mechanisms of sports injury in the foot and ankle occur on the playing field and with extreme, sudden force or impact of the joint and its surrounding soft tissue. For the ankle a mechanism of inversion versus eversion, dorsiflexion versus plantar flexion narrows the differential. Immediate swelling and non-weightbearing status after the injury are a concern. The anterior talofibular ligament is the most commonly injured structure in an acute ankle injury.

Symptoms

- Immediate swelling is a sign of ligamentous rupture or bony trauma because both of these injuries lead to subcutaneous bleeding. +++++ Blood typically accumulates more rapidly than other edema fluid.
- Inability to bear weight is a very important symptom +++++ because it is more indicative of bony injury.
- The majority of ankle injuries involve inversion. +++++ The patient's report of the location of the pain will most often be on the lateral ankle in this case.
- Patients may report the feeling or sound of a “pop” when a ligament or tendon is ruptured. ++++ Patients often report feeling like they were “shot” in the calf when an Achilles tendon rupture occurs. ++++

Signs

- Location of swelling should be noted (Table 6-1). Most often the swelling is laterally located. +++ Swelling across the anterior flexor crease of the ankle may be an indication of an intra-articular injury, like a talar dome fracture. ++++
- Ecchymosis is most often associated with ligamentous injury. +++++ Ligaments have a rich blood supply and partial or complete tear leads to a large amount of subcutaneous bleeding.
- The anterior drawer test is used to assess the integrity of the anterior talofibular ligament. +++++ It may reveal stability compared to the opposite side and laxity with an endpoint or laxity without a clear endpoint. It is important to compare with the uninjured side in special testing because preexisting laxity can be confused for acute ligamentous injury.

Table 6-1. Common Ankle Traumatic Injuries and Specific Signs

INJURY	COMMON SIGNS
First degree Inversion sprain	Swelling and point tenderness anterior to the distal fibula. Edema and bruising are generally mild. Stability should be present on anterior drawer and talar tilt.
Second degree Inversion sprain	Swelling and point tenderness as above are more severe. There may be laxity with an endpoint on anterior drawer and talar tilt.
Third degree Inversion sprain	This injury represents complete tear of the ATF. Swelling, tenderness and ecchymosis are severe. Anterior drawer and talar tilt reveal laxity without a clear endpoint.
High ankle sprain	Pain with dorsiflexion and eversion over syndesmosis. Positive squeeze test.
Peroneal tendon injury	Tenderness posterior to the lateral malleolus. The patient may experience difficulty with plantar flexion or eversion. Subluxation of the peroneal tendon over the lateral malleolus may be appreciated on palpation while the patient everts the foot.
Achilles tendon rupture	Tenderness over the Achilles. Inability to plantar flex when standing on one foot. Thompson test does not reveal plantar response.
Proximal fifth metatarsal fracture/Jones fracture	Pain over proximal fifth metatarsal.
Stress fracture	Metatarsal or calcaneus tenderness and pain.

- The talar tilt test also assesses the integrity of the lateral ankle ligamentous structures. +++++ Laxity and pain with testing compared with the opposite side indicate a higher grade of injury.
- The squeeze test involves compressing the tibiofibular axis. Pain with this maneuver suggests a rupture of the tibiofibular syndesmosis. +++++
- The Thompson test of the Achilles tendon involves squeezing the relaxed calf musculature with the knee in 90 degrees of flexion (patient kneeling on a chair) and noting for the presence of plantar flexion of the foot. +++++

Workup

- Radiography is indicated in the evaluation of ankle injuries, particularly if any of the following are present:
  - Pain on the posterior edge of the lateral malleolus
  - Pain on the posterior edge of the medial malleolus
  - If patient is totally unable to bear weight after the episode or for four steps in the office
  - Pain at the base of the fifth metatarsal
  - Pain at the navicular
- Mortise view should generally be included in an ankle x-ray series. Inversion stress views can also be obtained to determine the degree of joint opening, with more joint opening associated with complete ligamentous tear. MRI can detect ligamentous injury, but data on treatment favor functional treatment regardless of the degree of sprain.
- When a stress fracture is suspected, early radiography may be negative. Family physicians have the option to wait and repeat an x-ray in 1 to 2 weeks, at which point one may see signs of callus formation. Bone scan is more sensitive and reveals changes earlier or when no plain radiographic findings are apparent.

Comments and Treatment Considerations

Table 6-2. Treatment of Ankle Injuries

INJURY	TREATMENT
First degree Inversion sprain	Rest, ice, compression, elevation. Progressive weight bearing.
Second degree Inversion sprain	Rest, ice, compression, elevation. Crutches initially for comfort with progressive weightbearing. Early weightbearing yields better results. Medial, lateral stabilizing brace is indicated for 2 weeks continuously and up to 4 weeks with exercise. Patients should be taught range of motion, proprioception and strengthening exercises at the 2-week follow-up.
Third degree Inversion sprain	Rest, ice, compression, elevation. Crutches may be required. Functional treatment with progressive weightbearing and activity seem to be favored over immobilization. There is suggestion that surgical repair of rupture yields better outcomes than immobilization.
High ankle sprain	Rest, ice, compression, elevation. Non-weightbearing and orthopedic referral. This injury is often associated with higher-grade ankle sprain and even proximal fibular fracture.
Peroneal tendon injury	Rest, ice, compression, elevation. Non-weightbearing and referral to orthopedics.

Table 6-2. Treatment of Ankle Injuries—cont'd

INJURY	TREATMENT
Achilles tendon rupture	Often requires urgent surgical repair. Rest, ice, compression, elevation. Non-weightbearing and referral to orthopedics.
Proximal fifth	Rest, ice, compression, elevation. Fracture of the distal tip of the fifth metatarsal can be treated with 2-4 weeks in a hard-bottom shoe.
Metatarsal fracture/ Jones fracture	Rest, ice, compression, elevation. Fracture of the more distal portions of the metatarsal may require controlled ankle motion (CAM) walker immobilization and possible pinning.
Stress fracture	Rest, ice, compression, elevation. Non-weightbearing for 4-6 weeks and graded return to play are necessary.

KNEE TRAUMA

Knee trauma can occur on and off the playing field. The mechanism of injury will strongly lead the family physician in the direction of a correct diagnosis (Table 6-3).

Symptoms

- Immediate swelling is a symptom of ligamentous rupture or bony trauma because both of these injuries lead to subcutaneous bleeding. Blood typically accumulates more rapidly than other edema fluid. Effusions typically occur within 24 hours of injury, but especially in the first 4 to 6 hours. +++++
- Inability to bear weight is a very important symptom because it is more indicative of bony injury. ++++
- Patients may report the feeling or sound of a "pop" when a ligament or tendon is ruptured, especially in an anterior cruciate ligament (ACL) injury. ++++
- Typical ACL injury history involves foot planting and a twist. +++++
- Posterior cruciate ligament (PCL) is generally a dashboard injury. +++++
- Medial collateral ligament (MCL) and lateral collateral ligament (LCL) injuries often involve a blow to the opposite side of the knee from the affected ligament. +++++

Signs

- With ligament tear, the swelling is centrally located and hinders both knee flexion and extension. Ligaments have a rich blood supply and partial or complete tear leads to a large amount of subcutaneous bleeding or effusion.
- The Lachman test is used to assess the integrity of the ACL. It may reveal stability compared to the opposite side, laxity with

Table 6-3. Common Knee Traumatic Injuries and Specific Symptoms and Signs

INJURY	SYMPTOM	COMMON SIGNS
ACL tear	Pop with twisting Immediate swelling	Positive Lachman Pivot shift, anterior drawer
MCL tear/sprain	Pain on medial knee Lateral blow to the knee	First degree—negative valgus stress Second degree—positive stress but firm endpoint Third degree—positive stress without an endpoint
Meniscal tear	Medial/lateral twist, stress Locking/clicking sensation	Positive McMurray test with click Joint line tenderness and pain with squatting
PCL tear	Dashboard type injury	Positive posterior drawer
LCL sprain/tear	Lateral knee pain Medial blow to knee	First degree—negative varus stress Second degree—positive stress but firm endpoint Third degree—positive stress without an endpoint
Tibial plateau fracture	Axial loading and inability to bear weight	Knee effusion, positive x-ray

ACL, Anterior cruciate ligament; *LCL,* lateral collateral ligament; *MCL,* medial collateral ligament; *PCL,* posterior cruciate ligament.

an endpoint or laxity without a clear endpoint. It is important to compare to the uninjured side because preexisting laxity or history of ipsilateral knee surgery can be confused for acute ligamentous injury. The Lachman test eliminates the stabilization of the hamstring tendons and tends to isolate the ACL to a greater degree. It has a sensitivity of 86% and specificity of 91%. ++++

- The anterior drawer test of the knee may also assess for ACL injury using the same guidelines as above. It has a sensitivity of 20% and a specificity of 88%. The low sensitivity is due to patient guarding with the hamstring tendons or a PCL injury. ++++
- The pivot shift test assesses the ACL and has a sensitivity of 48% and specificity of 99%. +++++ Examiner skill is a determinant in the sensitivity of this test.
- The posterior drawer test assesses the integrity of the PCL. Again, laxity, endpoint, and comparison to the other knee should be noted. The sensitivity is 90% and specificity is 99%. +++++
- The McMurray test assesses the integrity of the medial and lateral meniscus. This test is performed in many different fashions; however, all methods involve a valgus stress with foot inversion and a varus stress with foot eversion while flexing and extending the knee. A positive test involves a palpable pop over the joint line of the affected side. The sensitivity is 26% and specificity is 94%. +++
- The combination of medial joint line tenderness (sensitivity 76%, specificity 29%) and painful squatting have good predictive value for meniscal injury. +++
- A valgus stress should be placed on the lateral knee to test the medial collateral ligament. A lateral blow to the knee is likely to yield a medial collateral ligament tear. The MCL and medial meniscus are continuous structures and are often injured together. +++++
- A varus stress should be placed on the medial knee to test the lateral collateral ligament. A medial blow to the knee may produce an LCL tear. ++++
- A patella apprehension test should be performed on a knee in which subluxation is suspected. A positive test is pain and laxity with a lateral stress to the medial patella. ++++

Workup

- Radiography is indicated in the evaluation of knee injuries, particularly if the following are present:
  - Patient is unable to bear weight immediately or in the office
  - Patient is tender over the patella
  - Patient is tender over the proximal fibular head
  - Patient is unable to flex knee past 90 degrees
  - Patient is more than 55 years old
- Order three x-ray views: the AP, lateral, and sunrise.
- A Hughston view may be substituted for the sunrise for a more specific view of the patella.

- If ligamentous or cartilage injury is suspected, an MRI of the knee without contrast may be ordered. Of note, although MRI is often ordered in the workup of ACL tear, MRI has not been shown superior to clinical examination in making this diagnosis.
- Wait 1 week for the knee effusion to decrease prior to ordering more studies and reexamine the knee when ROM is more accessible.

Comments and Treatment Considerations

Table 6-4. Treatment of Common Knee Injuries

INJURY	TREATMENT
ACL tear	Rest, ice, analgesics. For higher-grade tears, crutches and knee immobilization are often needed for patient comfort. Physical therapy is often initiated prior to surgery to maintain strength and range of motion. Consider referral to sports medicine or orthopedics within 1-2 weeks.
MCL tear/sprain	Relative rest, ice and analgesics. First degree: physical therapy and hinged medial/lateral stabilization brace Second degree: physical therapy and hinged medial/lateral stabilization brace Third degree: referral to sports medicine or orthopedics and possible surgery
Meniscal tear	Rest, ice, analgesics. Physical therapy versus surgical debridement or repair
PCL tear	Rest, ice, analgesics. Physical therapy and consideration of surgery
LCL sprain/tear	Rest, ice, analgesics. Physical therapy Surgical consideration if complete tear
Tibial plateau fracture	Immediate orthopedic referral

ACL, Anterior cruciate ligament; *LCL,* lateral collateral ligament; *MCL,* medial collateral ligament; *PCL,* posterior cruciate ligament

OSTEOARTHRITIS

Of all joint disorders worldwide, osteoarthritis (OA) occurs with the greatest frequency. OA is defined by joint pain, functional loss, and radiologic evidence of degeneration in the absence of significant systemic inflammatory response. "Wear and tear" is blamed for osteoarthritis and a specific injury is not always identifiable. Local inflammatory mediators do lead to articular degeneration, and local cytokines and interleukins do contribute to joint damage, but the disease is not one of systemic inflammation. Articular cartilage and subchondral bone are the tissue types most affected by OA. Predisposing

factors such as joint deformity, repetitive joint loading, trauma, and obesity do raise an individual patient's risk of developing OA.

Symptoms

- Joint pain +++++
- Stiffness, generally less pronounced after rest ++++
- A pattern of joint pain and/or stiffness over most days of a month ++++
- Swelling of the affected joint(s) ++++

Signs

- Patient age more than 38 to 40 years, depending on the joint affected. Younger patients tend not to have degenerative joint disease. +++
- Overweight or obese
- Angular deformity that may predispose to OA (i.e., genu valgum or varum)
- Effusion of joints where this finding is appreciable (knees).
- Bony enlargement.
  - Heberden's nodes (distal interphalangeal [DIP] joints of the fingers) +++
  - Bouchard's nodes (proximal interphalangeal [PIP] joints of the fingers) +++
- Limited active ROM secondary to stiffness or pain +++
- Limited passive ROM if effusion is present or if bony exostoses are prominent ++
- Crepitance or "crunching or grinding" feeling when joints are taken through range of motion ++
- Antalgic gait secondary to pain if knees or hips are affected

Workup

- Plain radiography may reveal typical patterns of OA. These changes may include osteophyte (bony spur) formation, joint space narrowing (often asymmetric), eburnation (increase in subchondral bone density), and subchondral cysts. The correlation of radiographic OA findings and arthroscopic disease is limited (correlation coefficient of 0.41 to 0.56 in one study of 125 patients). Additionally, bony spur formation may be present in asymptomatic individuals. Hence the need to use radiography in the context of appropriate history and physical examination. +++
- MRI is much better at detecting subtle changes in articular cartilage, but is not often needed to diagnose OA.
- Serum evaluation for inflammation using ESR typically reveals a normal value. +++++
- Joint fluid analysis typically reveals decreased viscosity and decreased fluid "stringing" and WBC count less than 2000/mL. +++++

Comments and Treatment Considerations

Lifestyle modification and nonpharmacologic interventions such as weight loss and exercise for muscle strengthening are very

important. Aerobic exercise is effective in improving gait, pain, and functional status. Ice massage may improve ROM, strength, and swelling in an osteoarthritic joint but the effect on pain is unproven. Heat does not appear to offer a benefit. Physical therapy and occupational therapy should be targeted at joint protection and strengthening of supporting muscles around the affected joint.

Pharmacologic interventions can also prove beneficial. Acetaminophen is the safest drug (in terms of GI events) used to treat OA in the absence of preexisting hepatic problems and can be used at the maximum dose of 4 g/day.

NSAIDs are more effective, though modestly, for improving pain, particularly in patients with moderate to severe disease. NSAID GI risk can be mitigated by the addition of a PPI.

COX-2 inhibitors may be considered for patients who cannot tolerate NSAIDs for renal or GI reasons. The GI risk reduction is actually greater with PPIs and NSAIDs, however, compared with COX-2 inhibitors. COX-2 inhibitors, additionally, may raise the risk of adverse cardiac events.

Tramadol and other opioid analgesics offer pain relief in OA. Tolerance, addiction, and side effects may limit the use of this class of medications.

Pain reduction can be achieved with injection of intra-articular steroid, particularly the knee. Triamcinolone appears to be superior to betamethasone. Functional improvement may not be achieved with intra-articular steroid.

Viscosupplementation gives pain relief comparable to steroid, but with a longer duration of effect. Brand-name products such as Hyalgan, Hylan G F 20, Orthovisc, and Synvisc have been studied in placebo-controlled trials and show benefit.

Joint fluid aspiration at the time of injection should generally be performed.

Semirigid bracing of joints and weight distribution change with shoe lifts are helpful for OA of the knee. Bracing of the first metacarpophalangeal (MCP) joint helps relieve pain.

Surgery for OA takes a number of forms. Arthroscopic debridement of damaged cartilage does provide relief, particularly for patients with cartilage damage that causes locking. Osteotomy repairs angular deformities that contribute to OA, but outcomes on the knee are not better than conservative therapy. Arthrodesis, or surgically decreasing joint mobility, helps with pain at the cost of ROM and is considered a salvage procedure in those who are not candidates for joint replacement.

Joint replacement relieves pain, although prosthesis failure with time is a concern. Typical life span of a prosthetic joint is 10 to 15 years.

Complementary and alternative measures including acupuncture offer relief from pain in OA, but studies are mixed in its superiority to sham acupuncture.

Certain brands (Rotta) of glucosamine are somewhat helpful for pain in OA, but can be expensive.

OVERUSE INJURIES

Chronic overuse tendon injuries are commonly mislabeled "tendinitis," whereas histologic findings demonstrate tendon degeneration without inflammation. As a result, the terms *tendinosus* or *tendinopathy* are more appropriate.

Tendons have a poor vascular supply proximal to their insertion, hence the osteotendinous junction is the most commonly affected site. However, these tendon injuries can occur at any level of the tendon.

The pathophysiology of tendinosis involves degeneration of highly arranged collagen fiber structures within the tendon. Disorientation of these fibers leads to tendon weakening and an increase of proteoglycan substance with neovascularization.

Radiographs can evaluate bony structures and joints, but films cannot be used to evaluate soft tissues. Films may demonstrate calcification at the osteotendinous junction. MRI is commonly used to evaluate soft tissues as is ultrasound, although it is highly operator dependent. MRI and ultrasound are commonly ordered for preoperative evaluation.

TENDINOPATHY

Symptoms

- May persist for up to 6 months
- Many patients respond more rapidly to nonoperative measures.

Comments and Treatment Considerations

Pain control, relative rest, ice, stretching, and eccentric strengthening are the mainstay of treatment. Analgesics such as acetaminophen or tramadol may be used. NSAIDs have a limited role because inflammation is often not present, although they may be beneficial during the acute stage.

Rest from the offending activity or sport prevents further tendon damage and may promote healing. Activities that do not cause pain symptoms may continue to be performed.

Cryotherapy has a potential benefit of analgesia, and may offer short-term relief. Prolonged application should be avoided to prevent injury to the skin. Its use should not exceed 15- to 20-minute intervals, and a moist towel should be used to avoid injury to the skin.

Stretching is widely accepted and considered beneficial.

Eccentric exercise involves lengthening of muscle fibers while the muscle is still contracting. These exercises stimulate collagen formation and aid in proper alignment of newly formed fibers.

Therapeutic ultrasound and massage are often used with varying results.

The use of corticosteroid injections is controversial. These injections aid in temporary relief of symptoms, but they can also weaken the tendon and ultimately lead to tendon rupture.

Braces are used to protect or unload the tendon during activity. Results vary, but braces are a safe treatment option.

Surgery is generally reserved for cases refractory to 6 months of nonoperative treatment. The procedure involves resection of the degenerative portion of the affected tendon.

ACHILLES TENDINOPATHY

Achilles tendinopathy is often seen in adult running athletes and its incidence increases with age. Causes include improper training techniques, sudden increase in activity, anatomic lower extremity deformities, uneven training surfaces, and poor-fitting shoes. Fluoroquinolone exposure has also been implicated in tendon degeneration and even rupture.

Symptoms

- Pain at the heel proximal to the tendon insertion +++
- Increase in pain associated with increased training schedule

Signs

- Tendon thickening and tenderness 2 to 6 cm proximal to its insertion ++++
- Frequently accompanied by foot alignment abnormalities (i.e., flat feet, heel varus, or foot pronation) +++
- Tightness of the calf muscles is often seen. +++
- Thomson's squeeze test (squeezing the gastrocnemius with the patient in the prone position) should be performed to verify that the tendon has not ruptured.

Workup

- The diagnosis is based primarily on physical examination and history.

Comments and Treatment Considerations

Eccentric or decline calf raises may be particularly helpful in this overuse injury. Stretching of the gastrocnemius-soleus complex and deep friction massage are often helpful.

LATERAL ELBOW TENDINOPATHY

Tendinopathy of the elbow can affect both the medial and lateral elbow. Lateral elbow tendinopathy is 10 times more common than its medial counterpart, and the patient's dominant arm is affected 75% of the time. Elbow tendinopathy more commonly occurs after age 40 +++, but may present at an earlier age.

Mechanisms causing lateral elbow tendinopathy, commonly mislabeled "lateral epicondylitis," include racquet sports ("tennis elbow") and repetitive activities that involve wrist extension or gripping.

Symptoms
- Insidious onset of dull or sharp pain in the lateral elbow +++++
- Typically affect the dominant arm +++
- Symptoms present with increased activity, and may appear at night in advanced stages.

Signs
- Tenderness at the lateral epicondyle, along the wrist extensor tendons, and the tendon insertion
- Resisted wrist extension commonly reproduces the patient's symptoms, especially with the elbow in extension.

Workup
- Lateral elbow tendinosis is a clinical diagnosis, but ultrasound and MRI are sometimes performed if treatment has failed.

Comments and Treatment Considerations
Tennis elbow brace or counterforce brace can sometimes be helpful. Equipment modification such as larger grip or vibration absorption devices may be useful. Corticosteroid injection provides short-term relief, although long-term outcomes are poorer in injection versus noninjection groups at 12 months.

MEDIAL ELBOW TENDINOPATHY

Medial elbow tendinopathy, commonly mislabeled "medial epicondylitis," can be seen in golfing ("golfer's elbow"), throwing sports, racquet sports, or activities that require repetitive wrist flexion.

Symptoms
- Insidious onset of dull or sharp pain in the medial elbow ++++
- More common in the dominant elbow
- Grip weakness
- Can be associated with nerve irritation in an ulnar distribution

Signs
- Tenderness along the medial epicondyle and tendons of the flexor pronator mass +++++
- Pain with resisted pronation and wrist flexion ++++
- Positive Tinel's testing (tapping on the nerve) posterior to the medial epicondyle suggests ulnar nerve irritation.
- Ulnar collateral ligament tear should also be suspected in throwing athletes. This can be assessed clinically with a valgus stress test of the elbow.

Workup
- The diagnosis is based primarily on physical examination and history.
- MRI or diagnostic ultrasound should be ordered if an ulnar collateral ligament tear is suspected.

Comments and Treatment Considerations

Corticosteroid injection should be used with caution because of the proximity of the lateral epicondyle to the ulnar groove and nerve. Injection into the nerve can cause damage.

ROTATOR CUFF TENDINOPATHY

Rotator cuff tendinopathy commonly presents with a history of repetitive overhead activities ++++, and this tendinopathy affects approximately 2% of the population. Incidence increases with age and is more common with overhead activities (i.e., throwing athletes, volleyball, and tennis) and in labor workers.

Pathophysiology is thought to involve repeat "impingement" or rubbing of the supraspinatus muscle with the coracoacromial arch during shoulder abduction. Tendon hypovascularity, which occurs approximately 2 cm from the distal supraspinatus insertion, also plays a role in this degenerative process.

Symptoms

- Pain at the region of the deltoid, which is worse with activity ++++
- Difficulty sleeping while lying on the affected shoulder ++++

Signs

- Positive *empty* can test (weakness/pain with resistance, arms 70 degrees abduction, 30 degrees forward flexion, and internal rotation with thumbs pointing down) +++
- Positive Hawkin's test (arm forward flexed 90 degrees and elbow flexed 90 degrees with progressive shoulder internal rotation) +++
- Positive Neer's test (fully pronated arm with maximum forward flexion) +++

Workup

- Radiographs: Radiographs may demonstrate spur formation of the acromion or tendon calcification.
- Ultrasonography: Diagnostic ultrasound can detect full-thickness rotator cuff tears, and can demonstrate chronic tendinopathy.
- MRI: Highly sensitive and specific for detection of rotator cuff tears and tendinopathy

Comments and Treatment Considerations

Avoidance of overhead activities in the acute phase, and patients should not be immobilized to prevent muscle atrophy. It is crucial to differentiate between rotator cuff tendinosis and tear. Tendinosis improves with rest, whereas tears often do not.

The goal of physical therapy is to reestablish pain free ROM through stretching exercises, and strengthen shoulder muscle once the patient has recovered from the acute phase.

Subacromial corticosteroid injections have shown to have a small benefit compared with placebo up to 1 month postinjection.

Subacromial decompression surgery involves shaving of the undersurface of the acromion to better accommodate the supraspinatus.

PATELLAR PROBLEMS

Dysfunction of the patellofemoral joint is the most common cause of anterior knee pain. It can be seen in all ages and professions but is especially frequent in athletes, and most specifically runners. Thirty percent of athletes experience anterior knee pain at some point during their competitive careers. Despite being a common problem, the specific etiology is frequently difficult to establish and is often multifactorial in nature. The patient history can be very helpful in suggesting a diagnosis, especially when a single traumatic event is involved and the mechanism of injury can be elucidated. However, frequently anterior knee pain is not associated with a specific traumatic event, but rather is insidious in onset. In these cases, a careful examination and appropriate use of diagnostic imaging tests are essential in establishing a diagnosis.

PATELLAR DISLOCATION

Acute, first-time patella dislocations are common in sports, and frequently occur in individuals younger than age 20. Studies have shown that the highest incidence occurs in women, ages 10 to 17 years. The mechanism can be a direct blow causing a valgus force across the knee, a direct medial blow to the patella, or with a twisting force associated with strong quadriceps contraction. Redislocation rates can be very high, approaching 60% in some studies.

Symptoms

- Sensation of the knee "popping" out of place +++++
- Anterior knee pain +++++
- Swelling +++++
- Medial bruising +++
- Instability or weakness +++
- Reluctance to bear weight

Signs

- If seen acutely the knee will typically be held in a flexed position with the patella prominent along the anterolateral side of the knee. +++++
- Large effusion/hemarthrosis +++++
- Tenderness along the medial retinaculum, often with ecchymosis in this area ++++
- May be marked hypermobility of the patella ++
- Apprehension when the patella is displaced laterally +++++

Workup

- Thorough knee examination looking for other associated injuries such as disruption of the ACL, MCL, or menisci.
- All suspected patella dislocation should have a radiograph to evaluate for associated osteochondral injury.
- Evaluation of possible predisposing anatomic factors:
  - Increased Q angle
  - Trochlear dysplasia
  - High-hiding patella
  - Vastus medialis obliquus (VMO) weakness or atrophy
  - Increased femoral anteversion
  - External tibial torsion
  - Pes planus or foot hyperpronation
  - Genu valgum

Comments and Treatment Considerations

If seen in the acute setting, the patella can often be easily relocated by extending the knee with gentle lateral to medial pressure on the patella. The examiner may hear or feel a "clunk" as the patella is reduced.

If a large hemarthrosis is present, aspiration under sterile techniques can often provide some symptomatic relief and allow improved ROM. Presence of fat globules in the aspirated blood can indicate an associated fracture.

Apply ice to minimize swelling. NSAIDs provide pain reduction.

It is generally accepted that a short period (2 to 3 weeks) of immobilization with the knee in extension may allow disrupted structures to heal and minimize redislocation rates. Early physical therapy will also help.

Return to sports when full ROM and strength are regained, without symptoms of instability. This may take 6 to 12 weeks. Consider surgery for recurrent dislocations.

PATELLAR TENDINOSIS (TENDINITIS)

Tendinopathy of the patella tendon is an overuse condition resulting from repetitive loading of the knee extensor mechanism. This loading typically occurs with jumping, and therefore the condition has been termed "jumper's knee." It is believed that repetitive stresses cause microdamage, and tendon fiber failure, without allowing adequate time for tendon healing.

Symptoms

- Well-localized pain typically at the inferior pole of the patella ++++
- Pain begins gradually and may only be present during intense physical activity or after activity. As symptoms persist, the pain becomes present during normal daily activities and even at rest. +++++
- Pain may be intensified with rising from squatting position, prolonged sitting with bent knees, and the use of stairs. ++++

Signs
- Tenderness at the inferior pole of the patella, and upper portion of the patella tendon when the knee is in full extension ++++
- Pain may be elicited with repetitive squats. +++
- Palpable thickening of the tendon compared with the contralateral side ++
- Atrophy of the quadriceps musculature ++

Workup
- History and clinical examination should be used to differentiate patella tendinosis from patellofemoral pain.
- Careful questioning as to potential aggravating activities
- The only anatomic factor shown prospectively to be predisposing is tight musculature, particularly the quadriceps muscles.
- X-rays may be helpful to differentiate patella tendinosis from other conditions such as patellofemoral OA.

Comments and Treatment Considerations
Relative rest and avoiding all potentially aggravating activities are the key features of treatment. Despite this being a relatively non-inflammatory condition there is still debate as to whether inflammatory mediators such as COX play a role in its pathogenesis, so NSAIDs may still have a role in a treatment regimen.

Ice can be used as an analgesic. It may also reduce the neovascularization that histologically has been shown to occur, and may play a role in the pathogenesis.

Eccentric exercise is most helpful for tendinopathy.

Deep tissue massage has shown some promise in animal studies.

The length of time to recover is generally related to the severity of the disease progression. It is not unusual for full recovery to take 4 to 6 months. Conservative treatment has been shown to be equal to surgical treatment in terms of length of time to recovery, and outcomes. However, cases not responding to a prolonged, intensive course of conservative treatment may be considered possible candidates for surgery.

PREPATELLA BURSITIS

The prepatella bursa is a fluid-filled sac lying between the patella and the skin. The main function of the bursa is to provide cushioning against external pressure over the patella, such as occurs with kneeling. Inflammation of the bursa can occur from a single traumatic event, from repetitive pressure on the patella, or rarely from an infection. This condition is frequently seen in professions that involve kneeling such as carpet layers, tilers, gardeners, and roofers.

Symptoms
- Anterior knee swelling +++++
- Anterior knee pain especially when pressure is placed over the patella such as with kneeling ++++
- Occasionally may have some surrounding skin erythema ++

Signs
- Enlarged, boggy bursa on palpation ++++
- Bursa may be tender to the touch. +++
- May lack full flexion due to skin tension around the inflamed bursa ++
- May be palpable crepitance ++
- Occasional surrounding skin erythema, warmth, or cellulitis ++

Workup
- Aspiration of the bursa can be performed under sterile conditions if the bursa is very tense and limiting full joint motion, or if infection is suspected.
- X-rays should be performed in cases that occur from single traumatic fall or blow to the patella to rule out a fracture.

Comments and Treatment Considerations
Ice and NSAIDs: If a bursa aspiration is performed, a compression wrap should then be applied, followed by a short period (1 to 2 days) of immobilization. Aspiration in chronic bursitis is usually not helpful.

In chronic bursitis the goal is to avoid inciting activities or use protective knee pads. If a septic bursa is suspected the bursa should be aspirated, and fluid sent for analysis. Mild cases may be treated with oral antibiotics, but more severe cases may need IV antibiotics, bursal lavage, or incision and drainage. The typical bacterial causes are *S. aureus* and group A β-hemolytic *Streptococcus*.

Surgery to remove the bursa is elective, and is typically performed if there is an excessively large, chronically inflamed bursa that extends beyond the margins of the patella.

PATELLOFEMORAL PAIN SYNDROME

Patellofemoral pain syndrome is a term used to describe anterior knee pain that seems to lack a clear-cut anatomic or pathologic cause. Usually this diagnosis is considered when other conditions have been ruled out by history, examination, and radiographic evaluation. The exact origin of the pain in patellofemoral pain syndrome is unclear. It is theorized that the pain may originate from the subchondral bone, or surrounding soft tissue structures and the cause is likely multifactorial, hence the wide variety of treatment options that have been recommended.

Symptoms
- Dull, achy anterior knee pain that can be described in a diffuse area centered around the patella, or localized very specifically to the patella ++++
- Insidious in onset +++++

- Knee may be described as "stiff," or the pain intensified after prolonged sitting with the knee in a flexed position ++++
- Occasional sense of instability when the pain is intense ++

Signs

- Absence of effusion and stable ligament examination ++++
- May have a positive patella grind test in which compression of the patella with the knee in extension reproduces pain +++
- May have tenderness along the medial and lateral facets of the patella +++

Workup

- X-rays to rule out other causes such as advanced patellofemoral OA and to assess the bony anatomy of the patellofemoral joint
- Complete evaluation of the lower extremity mechanics including the hip, ankle, foot, and general analysis of gait
- Consider an MRI if the diagnosis is in doubt, or if the patient fails to respond to appropriate treatment. An MRI is useful in visualizing the articular cartilage and surrounding structures.

Comments and Treatment Considerations

NSAIDs may have a benefit in short-term pain relief. Conservative treatment is favored over surgical treatment unless there is correctable pathology identified, or severe alignment issues.

Treatment should be tailored toward the individual patient, emphasizing correction of any potential correctable anatomic contributors. Physical therapy has been shown in several studies to be beneficial in reducing pain in patellofemoral pain syndrome, but not necessarily in improving function. The type of therapy likely does not matter because both open and closed kinetic chain exercises have shown equal benefit. There are no data to suggest that any exercises are able to isolate and strengthen the VMO, which is essential in dynamic medial stabilization of the patella, so a generalized program aimed at lower extremity flexibility, and strengthening is adequate.

Patella taping or McConnell taping, which attempts to medially shift the patella more central into the trochlea, has been used in the treatment of patellofemoral pain syndrome. In uncontrolled trials, taping has demonstrated some benefit.

Bracing has shown no benefit.

Foot orthoses may be beneficial especially in those individuals with abnormal foot mechanics, but very few studies exist to prove efficacy.

Acupuncture has shown some benefit in reducing pain, but no placebo-controlled trials exist.

Other treatment modalities which have not shown benefit include ultrasound, chiropractic manipulation, electromyelogram (EMG) biofeedback, and low-level laser treatment. Body weight plays a significant role in stresses across the patella, so weight loss is beneficial in any patient who is overweight and has patellofemoral pain syndrome.

POLYMYALGIA RHEUMATICA

Polymyalgia rheumatica (PMR) and giant cell arteritis (GCA), or temporal arteritis, are closely related conditions. The pathogenesis of these diseases is poorly understood but a "multihit" phenomenon has been proposed with possible link to infectious agents. PMR is quite common (1/133) among certain studied populations (Olmstead County, Minnesota) to an incidence of 50/100,000 in more generalized populations in the United States. GCA incidence is between 17 and 50/100,000. Comparatively, OA affects between 42 and 296/1000 people, depending on age and sex. Fifteen percent of PMR cases are associated with GCA.

Symptoms

- Bilateral aching and stiffness for 1 month or more and involving two of the following areas: neck or torso, shoulders or proximal regions of the arms, and hips or proximal aspects of the thighs +++++
- Some patients recount difficulty initiating movement after rest known as "gelling phenomenon."
- Shoulder pain (in 75% to 90% of patients) radiating toward the elbows ++++
- Hip and neck pain are present in 50% to 70% of patients, with hip pain radiating toward the knees. +++
- Distal asymmetric join pain is present in half of patients. +++

Signs

- Patient older than 50 years +++++
- Objective physical examination findings are often out of proportion to the patient's report of pain. It is uncommon to find swelling or tenderness on joint examination. ++++
- Patients report pain with both active and passive ROM. ++++
- Asymmetric knee, hand/wrist, and dorsal foot pitting edema in some of those who have distal arthritis

Workup

- Plain radiographic imaging may be used to rule out other diagnoses such as occult fracture, calcific tendinitis, and primary or metastatic neoplasm, though it is not needed.
- MRI or ultrasonography of the shoulders can be used to detect subacromial or subdeltoid bursitis, which can be present in PMR.
- Nonspecific rheumatologic screening tests (rheumatoid factor ANA) are not useful except in ruling out other polyarthritides. Evaluation for hypothyroidism with TSH and CBC to assess for hematologic causes of weakness and fatigue helps rule out other diagnoses.
- ESR is elevated greater than 40 mm/hr. +++++
- To make the diagnosis or PMR by established criteria, all of the following must be present: patient older than 50, the joint pain

and stiffness mentioned previously, ESR greater than 40 mm/hr, and exclusion of other diseases. One diagnostic criterion set also includes response to low-dose prednisone as necessary.
- ESR measurement can be used to monitor the activity of the disease, but symptoms should guide dosing of steroids.

Comments and Treatment Considerations

NSAIDs, ice, heat, massage, and physical therapy are not useful in relieving symptoms of PMR.

The primary treatment of PMR is administration of low-dose steroids (equivalent to 10 to 20 mg of prednisone by mouth daily). Steroid should be continued for 2 to 4 weeks and then withdrawn slowly to prevent relapse. Fifty percent of patients do have spontaneous relapse of symptoms.

A randomized, double-blind controlled trial has demonstrated that the addition of methotrexate to prednisone results in steroid sparing and shorter courses of steroids. This regimen might be administered in cooperation with a consulting rheumatologist.

Complications of steroids must be attended to. In particular, patients on longer-term steroids should be treated with calcium and vitamin D and monitored for the development of osteoporosis.

RHEUMATOID ARTHRITIS

Rheumatoid arthritis (RA) is the most common inflammatory arthritis. It is an appendicular arthritis, meaning that it preferentially affects the peripheral joints and skeleton as opposed to the central skeleton. The primary target of the inflammation is the synovium and joint cartilage. The erosion and destruction of cartilage and bone can rapidly lead to joint deformities, functional impairment, and disability. Worldwide prevalence of RA is approximately 1%. Women are affected two to three times more frequently than men and incidence peaks between the fourth and sixth decades of life.

Symptoms

- Onset is insidious (over weeks to months) in 55% to 70% of patients +++; subacute (over days to weeks) in 15% to 20%, ++ or acute (over a few days) in 8% to 15%. ++
- Joint pain is polyarticular in two thirds of patients. ++++ Symmetry is an important diagnostic feature. ++++ Pain is most often in the small joints of the hands and feet, in particular the wrists and PIP, MCP, and MTP joints.
- The presence and duration of morning stiffness are useful in diagnosis and in following the clinical course of the disease. Morning stiffness lasting greater than 60 minutes strongly suggests inflammatory arthritis. ++++
- Systemic symptoms may include fatigue, malaise, weakness, low-grade fever, and weight loss. ++

Signs

- Characteristic involvement of small joints of hands and feet ++++
- RA often affects larger joints such as the elbows, knees, shoulders, and cervical spine.
- DIP joints, sacroiliac joints, and the spine are rarely affected.
- The key physical finding is synovitis, warmth, bogginess, and tenderness around the joints affected. ++++ Joints may be red. ++++ The swelling is a result of both effusion within the joint and synovial proliferation.
- Limitation of active and passive ROM ++++
- Weakness may be out of proportion to pain.
- Late findings:
  - Joint deformity such as radial deviation of the wrists and ulnar deviation at the MCP joints.
  - Swan-neck deformity (extension at PIP, flexion at DIP) and boutonniere deformity (flexion at PIP, extension at DIP) result from tendon rupture and subluxation.
  - Joint instability
  - Rheumatoid nodules are present in 15% to 25% and occur over extensor surfaces and areas under pressure. Nodules may develop abruptly or gradually. ++

Workup

There is no single test to diagnose RA—its diagnosis is primarily clinical. The disease needs to present for at least several weeks to make a diagnosis. The American College of Rheumatology (ACR) criteria require four out of a possible seven criteria that provides a sensitivity and specificity of 90%.

- Morning stiffness that lasts longer than 1 hour (present for >6 weeks).
- Arthritis of three or more joint areas (>6 weeks)
- Arthritis of hand joints (wrist, MCP, PIP) (>6 weeks)
- Symmetric arthritis (present for >6 weeks)
- Rheumatoid nodules
- Serum rheumatoid factor positive
- Radiographic changes (erosions or periarticular osteopenia)

Careful observation of disease activity over time can also help to establish the diagnosis.

The differential diagnosis includes systemic lupus erythematosus, psoriatic arthritis, arthritis associated with viruses such as parvovirus, hepatitis B and C, and seronegative spondyloarthropathies.

Baseline lab studies recommended by the ACR subcommittee of RA include:

- Complete blood count (CBC) may reveal anemia in 80% ++++ of patients, which is usually normochromic, normocytic with thrombocytosis (platelets are an acute phase reactant and typically increase with systemic inflammation).
- Rheumatoid factor is present in 70% to 85% of patients. ++++ High titers correlate with more severe disease, the presence of

rheumatoid nodules, and extra-articular manifestations of RA. The titer does not have prognostic value in individual patients and serial titers are not useful. False positives are seen in numerous conditions including viral illnesses such as mononucleosis, bacterial infections such as spontaneous bacterial endocarditis, and other chronic inflammatory conditions such as sarcoidosis.

- ESR or CRP is elevated in 90%. ++++ These tests can correlate with the degree of synovial inflammation but it varies greatly from patient to patient. The tests can be a useful objective measure to follow clinical response.
- Baseline renal and hepatic function can be useful to establish a baseline before starting medications with potential renal or hepatic toxicity.

Other tests:

- ANA is positive in 30% of RA patients. ++ Positive results are not specific to RA and the presence of ANA does not rule rheumatoid arthritis in or out.
- Anti-citrullinated peptide (CCP) antibodies are present in about 80% of RA patients. ++++ The anti-CCP2 test has equal sensitivity to rheumatoid factor (RF) test, but far better specificity (90% to 98%).
- Synovial fluid reveals 2000 to 20,000 leukocytes per mL, with 50% to 80% being neutrophils.
- X-ray reveals joint space narrowing that is symmetric, bony erosions, osteopenia, resorption of ulnar styloid, C1-2 subluxation, and instability. Radiographic evidence of erosions becomes apparent after the disease has been present for at least several months.

Comments and Treatment Considerations

Complications of rheumatoid arthritis can affect almost every body system:

- Hematologic: Includes anemia, +++ increased rate of lymphoproliferative diseases including Hodgkin's and non-Hodgkin's lymphoma, multiple myeloma, and leukemia
- Cardiac: Pericarditis (rare clinically but 50% at autopsy, +++), myocarditis, increased rates of coronary artery disease
- Pulmonary: Interstitial fibrosis, rheumatoid nodules
- Ophthalmologic: Keratoconjunctivitis, sicca syndrome, ++ episcleritis and scleritis
- Vascular: Vasculitis
- Neurologic: Myelopathy from cervical spine instability, peripheral nerve impingement
- Renal: Drug toxicity can lead to renal impairment.
- Felty syndrome: Triad of RA, splenomegaly, and neutropenia. These patients are at increased risk of serious bacterial infections of the lungs, skin, and perianal areas.
- RA is treated with a “reverse pyramid” approach in which disease modifying antirheumatic drugs (DMARDs) are begun early. This is done because joint destruction begins within weeks of symptom onset and DMARDs have significant benefits when used early. It is

therefore of critical importance to recognize and diagnose RA so that treatment can begin as rapidly as possible.

The goals of therapy are to relieve pain, reduce inflammation, improve function, prevent complications, and resolve the pathogenic process. There is no evidence that any medical therapy can heal erosions, reverse joint deformity, or cure RA.

The categories of current pharmacologic treatment are:

- NSAIDs/COX-2 inhibitors: Relieve symptoms of pain and swelling but do not alter disease course. There is no one consistently superior NSAID for RA.
- Glucocorticoids: Highly effective at relieving symptoms and can slow joint damage. Side effects of this class of medications are severe, so glucocorticoids are most often used as a bridge therapy until DMARDs take effect.
- DMARDs: Include sulfasalazine, hydroxychloroquine, methotrexate, and leflunomide. This class includes immunosuppressives such as azathioprine and cyclosporine.
- Biologic response modifiers: Infliximab, adalimumab, rituximab, etanercept, abatacept, anakinra.

Generally speaking, early referral to rheumatology is advisable to speed access to disease-modifying drugs. There is increasing evidence that combinations of DMARDs may be more effective than single-drug regimens. There is no clear evidence for dietary interventions such as supplemental fatty acids and vegetarian diets.

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